NEUROPATHIC ULCERS

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NEUROPATHIC ULCER OVER PROMINENT FIRST METATARSAL HEAD

A 54-year-old male patient with type 2 diabetes diagnosed at the age of 45 years was referred to the outpatient diabetic foot clinic because he had developed an ulcer on the plantar area of his left foot. He was treated with antidiabetic tablets and diabetes control was good (HBA_{1c}: 7.1%). On examination he had a full thickness ulcer on the head of the first metatarsal in an area where there was gross callus formation (Figure 5.1). No signs of infection were observed. He had findings of diabetic neuropathy (no sensation of pain, light touch, temperature or vibration). Vibration perception threshold was 45 V on the right and >50 V on the left foot. His peripheral pulses were palpable and the ankle brachial pressure index was 1.2 bilaterally.

The patient did not have a previous history of problems with his feet. He denied any pain or trauma. He was aware of the presence of the ulcer, after he had seen discharge on his socks and the insole of his shoes. Debridement of the ulcer was carried out and the patient was advised to rest his feet; therapeutic footwear was also prescribed (Figure 5.2). This patient attended the diabetic foot clinic on a weekly basis and he changed the dressings every day. The ulcer healed completely in 10 weeks.

This is a typical neuropathic ulcer. Such ulcers are painless — unless they become infected — and develop in patients with neuropathy under areas of high-pressure loading. A callus forms at points of high repetitive pressure on the sole of the foot

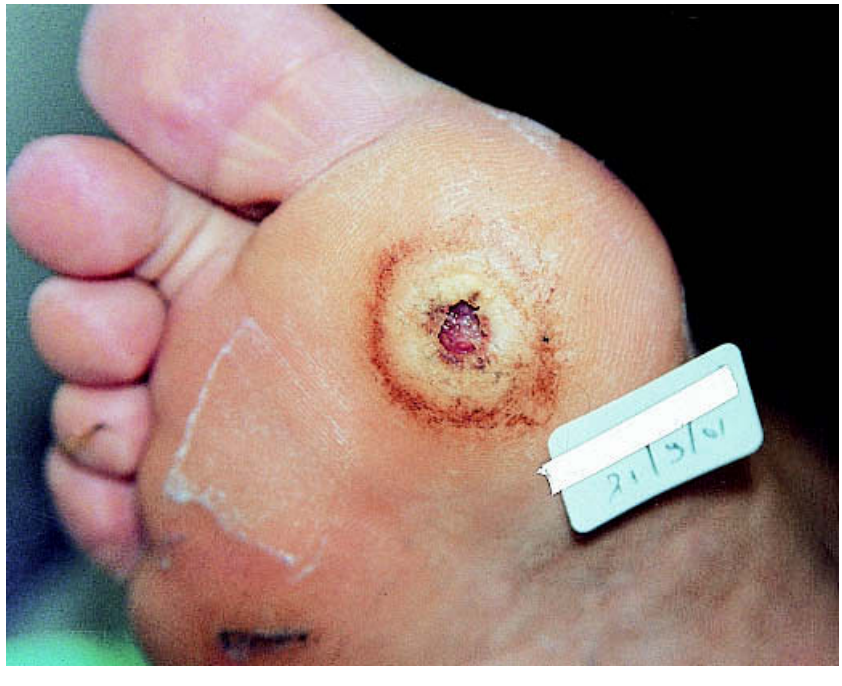

Figure 5.1 Neuropathic ulcer on the first metatarsal head

Figure 5.2 Therapeutic footwear prescribed for the patient whose foot is shown in Figure 5.1. Among the most commonly used therapeutic footwear is the rocker style shoe. Notice that the apex (ridge) of the rocker sole is located behind the metatarsal heads

and is a powerful predictor of ulceration. Such areas are the metatarsal heads and the plantar aspect of the great toe. Callus formation on the heel is not very common. In addition, calluses can develop over areas of bony prominences at other sites in the case of foot deformities (claw and hammer toes, toe overriding, neuro-osteoarthropathy). Even though the etiology of callus formation has not been determined, the fact that a callus acts as a foreign body in the shoe and contributes to high plantar pressure is well known. It is therefore recommended that callus formation should be prevented and when a callus is present, it should be removed regularly. Appropriate footwear is thought to prevent callus formation and the efficacy of this measure will be reflected by the proportion of patients wearing the correct footwear who develop ulcers. Hemorrhage into a callus is known as a 'pre-ulcer' and it should be treated as an ulcer.

Keywords: Neuropathic foot ulcer

NEUROPATHIC ULCER OVER PROMINENT METATARSAL HEADS

A 53-year-old female patient who had had type 2 diabetes since the age of 41 years and was being treated with insulin, was referred to the outpatient foot clinic because of a chronic foot ulcer. She had background retinopathy, cataract, hypertension and ischemic heart disease. The patient complained of numbness and a sensation of pins and needles in her feet, which worsened during the night.

On examination she was found to have a full thickness ulcer under her second and third prominent metatarsal heads and claw toes (Figure 5.3). The patient had severe peripheral neuropathy (no sensation of light

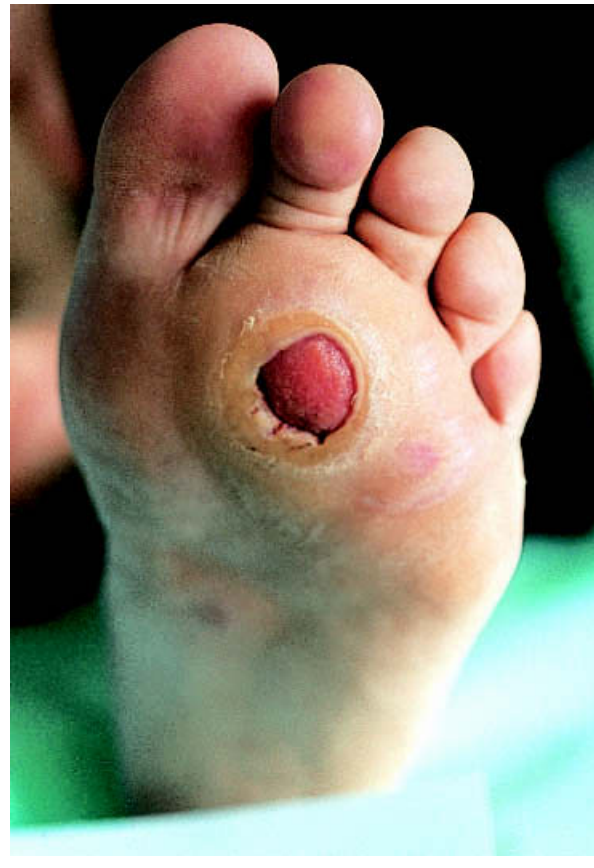

Figure 5.3 Neuropathic ulcer over prominent metatarsal heads

touch, pin prick, temperature, 5.07 monofilament, absence of Achilles tendon reflexes; and a vibration perception threshold over 50 V). Peripheral pulses were palpable and the ankle brachial pressure index was 1.1 bilaterally.

The patient reported having a callus — probably due to high peak plantar pressures at the site of the callus — for the past 2 years, which she treated with pumice stone. Six months before her first visit, she noticed that the callus was harder and its base had become purple; when she decided to remove it using a blade, an ulcer developed, which she then treated with local antiseptics.

Debridement of the ulcer was carried out on a weekly basis.

Healthy granulating tissue was present at the base of this clean ulcer, together with mild callus formation at the border. The patient was advised to take prolonged bed rest and the ulcer healed completely in 6 weeks. Appropriate preventive footwear and orthotic insoles were prescribed in order to prevent the formation of a new ulcer.

This patient erroneously thought that pain in her feet was proof of a healthy peripheral nerve system. The combination of painful neuropathic symptoms and at the same time, complete absence of sensation

(a 'painful–painless foot') is a quite common feature of neuropathic diabetes.

Keywords: Neuropathic ulcer; granulating tissue

NEUROPATHIC ULCER OVER A COLLAPSED MIDFOOT

A typical neuropathic ulcer under a bony prominence in a patient with midfoot collapse due to neuro-osteoarthropathy is shown in Figure 5.4. Callus formation is present at the margins of the ulcer, while its base is clean, covered by healthy granulating tissue.

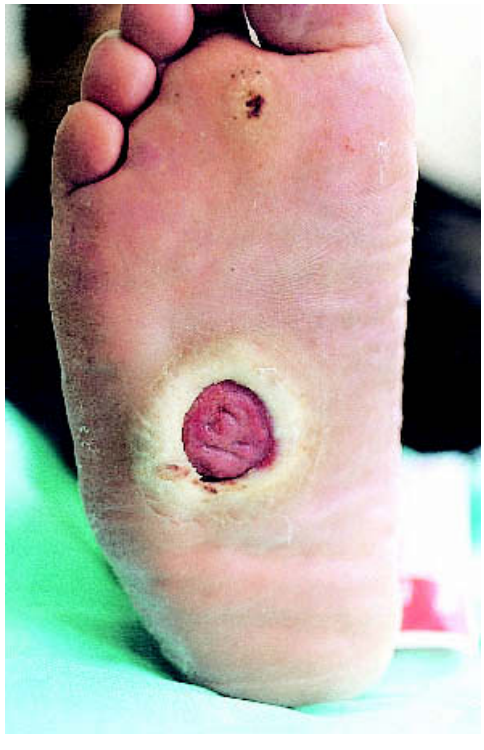

Figure 5.4 Neuropathic ulcer over a bony prominence in a patient with neuro-osteoarthropathy

Therapeutic footwear was prescribed (extra depth shoes with an orthotic insole and a window under the ulcerated area) and the patient was advised to minimize his activities. The ulcer healed in 3 months.

Ulcers in patients with midfoot collapse recur very often. Prevention of new ulcers over the same bony prominence is achieved by prophylactic surgery (osteotomy of the prominent bone). Preservation of plantar ligaments is essential, since their extensive resection may cause progression of the rocker bottom deformity.

Keywords: Neuropathic ulcer; bony prominence; prophylactic osteotomy

NEUROPATHIC ULCER UNDER FOURTH METATARSAL HEAD

A 74-year-old female patient with type 2 diabetes diagnosed at the age of 62 years, was referred to the outpatient diabetic foot clinic because of callus formation on her right sole. She was being treated with insulin and had a history of hypertension and ischemic heart disease.

On examination she was found to have severe peripheral neuropathy and normal peripheral pulses. In addition, significant muscle atrophy of her feet, claw toes and a hemorrhagic callus on the fourth metatarsal head of her right foot were found (Figure 5.5). An impressive finding was the palpation of her metatarsal heads just below the skin as the fat pads had been displaced anteriorly. After callus removal a superficial ulcer was revealed (Figure 5.6). An anteroposterior radiograph showed diffuse

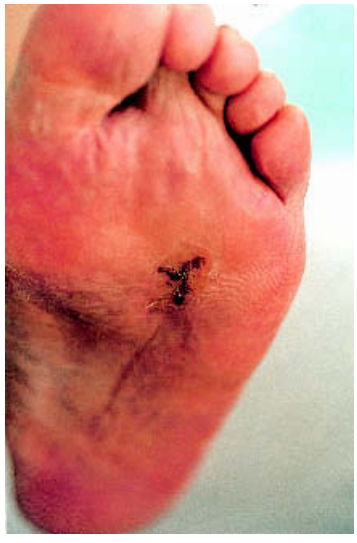

Figure 5.5 Hemorrhagic callus under the fourth metatarsal head. Claw toes and prominent metatarsal heads are also present

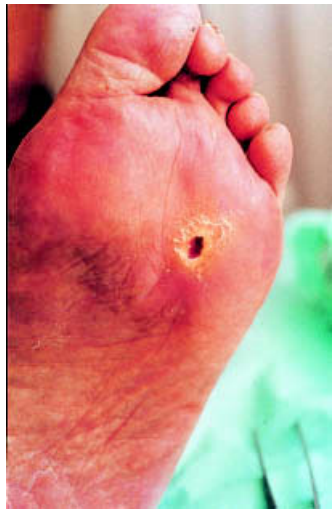

Figure 5.6 A neuropathic ulcer in the same patient whose foot is shown in Figure 5.5

demineralization of the foot and significant widening with periosteal reaction at the metatarsal heads (Figures 5.7 and 5.8). The patient was advised to rest. Extra depth

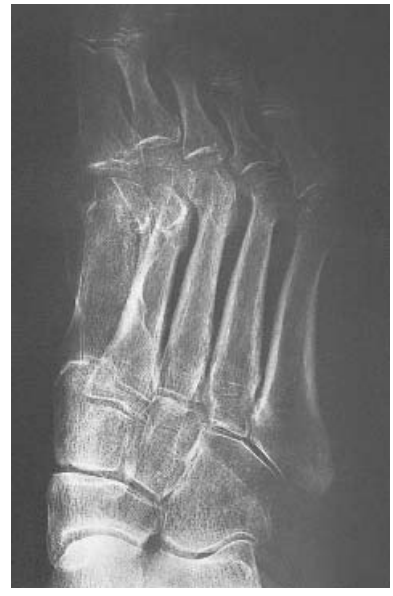

Figure 5.7 Diffuse osteopenia and significant widening with periosteal reaction on the metatarsal heads can be seen in this X-ray of the foot shown in Figure 5.5

shoes and orthotic insoles were prescribed in order to accommodate her deformed toes and relieve the load under the metatarsal heads. Post-debridement in-shoe pressures when she used her own shoes showed a significant load under her metatarsal heads (Figure 5.9 Panel A). The maximum pressure in this area was 282 kPa; however, after insertion of an orthotic insole the maximum in-shoe pressure was reduced to 155 kPa (Figure 5.9 Panel B). The ulcer healed in 8 weeks.

Reduced thickness of the fat pad is associated with high plantar pressures. Although some authors have suggested that threshold pressures of 500–1000 kPa may lead to the development of foot ulceration when walking barefoot, it seems that each patient has an individual threshold. In the present case the maximum pressure was obviously below this threshold. However, high plantar

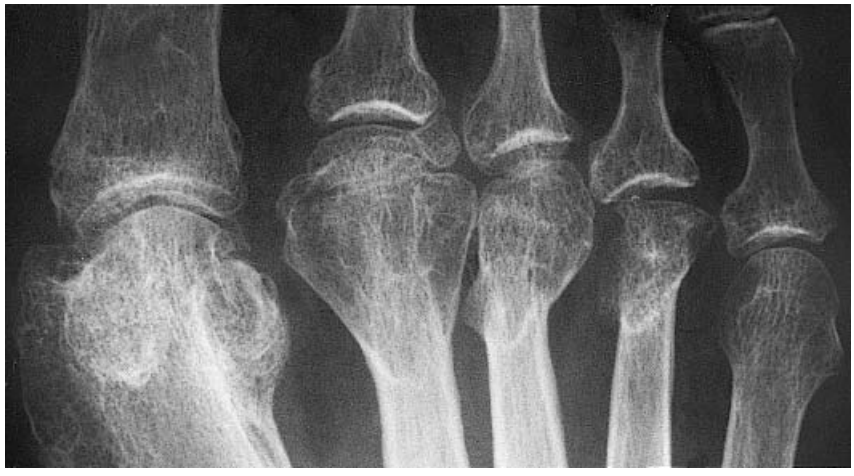

Figure 5.8 Significant widening with periosteal reaction of the first three metatarsal heads (same patient whose foot is shown in Figures 5.5–5.7)

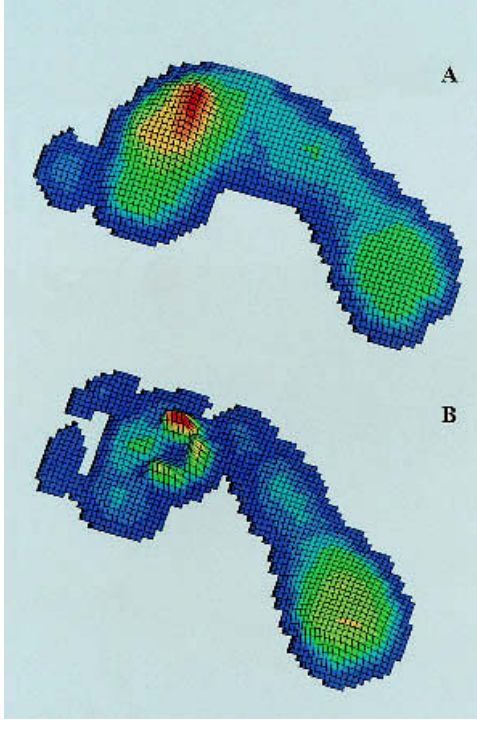

Figure 5.9 Plantar pressures before (A) and after (B) orthotic insoles in the patient whose foot is shown in Figures 5.5–5.7

pressures alone do not cause foot ulceration; a combination of different risk factors (mentioned in Chapter 1) is necessary for the development of ulceration.

Demineralization of the foot bones is not common, but when this occurs it signifies an adequate circulation, which is a prerequisite for bone resorption. Localized, mature periosteal reaction and demineralization involving metatarsal heads is common in diabetic patients with neuropathy. Its etiology is poorly understood. Focal osteolysis of phalanges, metatarsal heads, and other single foot bones, as well as stress fractures of the metatarsal heads can also be seen in neuropathic patients. Bone resorption at the phalanges may be so extensive that a part or even a whole phalanx may be resorbed. Metatarsal resorption usually starts from the metaphysis and extends to the epiphysis sparing the diaphysis. Bones which have become demineralized may have a pencil-like appearance.

Keywords: Neuropathic ulcer; plantar pressures, periosteal reaction

NEUROPATHIC ULCERS UNDER PROMINENT METATARSAL HEADS

This 32-year-old type 1 female diabetic patient, diagnosed at the age of 16 years, attended the outpatient diabetic foot clinic for chronic neuropathic ulcers of her feet. She was treated with intensive insulin treatment. The patient had a renal transplant at the age of 30 years, because of end-stage renal failure due to diabetes, and she had laser treatment on both eyes at the age of 28 years. Soon after her transplantation she noticed a bulla under her last three left metatarsal heads which readily ruptured and a superficial ulcer developed. She also reported an ulcer of 2 years' duration under the third metatarsal head of her right foot. She had never been instructed in foot care and had never worn the correct footwear. She had two small children and had not been taking good care of her feet. The patient was being treated with erythropoietin injections, cyclosporin, methylprednisolone, mycofenolate mofetil and furosemide.

On examination she was found to have bounding pedal pulses, and severe diabetic neuropathy. The vibration perception threshold was above 50 V in both feet bilaterally.

A non-infected neuropathic ulcer was noted under her left third, fourth and fifth metatarsal heads. Its dimensions were 3.5 × 4 × 0.4 cm, and it was surrounded by callus. A smaller neuropathic ulcer was also observed under her midsole (Figure 5.10). Claw toe deformity of her lesser toes, dry skin and desquamation of the tip of her third toe were also present. Under her

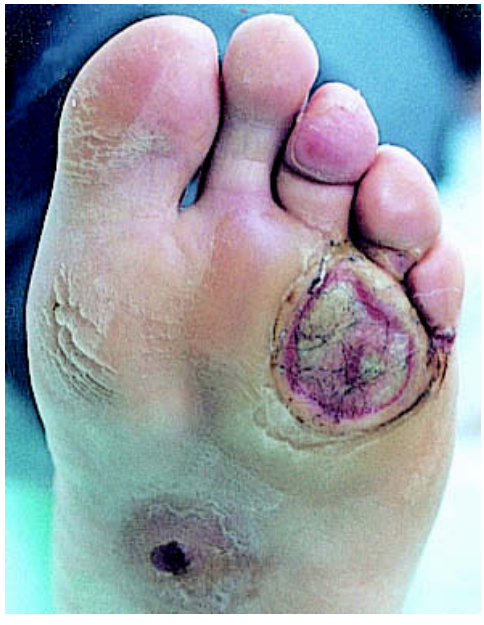

Figure 5.10 Neuropathic ulcers under prominent metatarsal heads and on the midsole. Claw toes and dry skin are also apparent

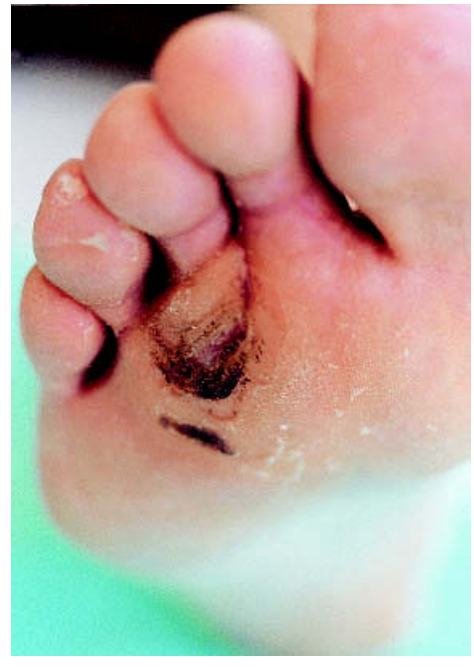

Figure 5.11 Neuropathic ulcer surrounded by callus. Claw toe. Right foot of patient whose left foot is shown in Figure 5.10

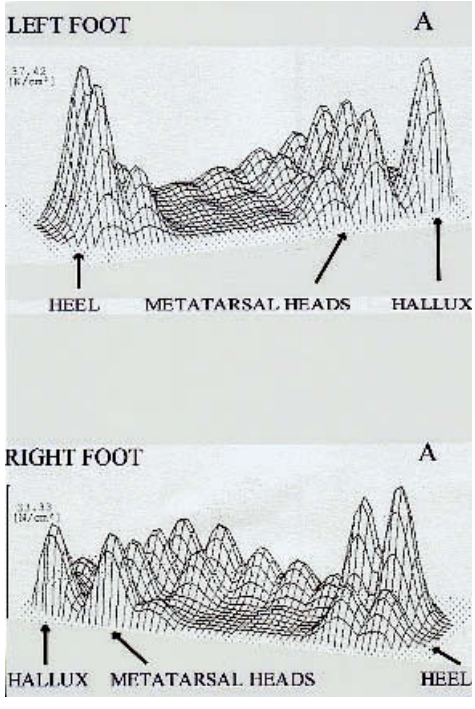

Figure 5.12 Original in-shoe peak plantar pressures on the left (upper panel) and right foot (lower panel) of the patient whose feet are illustrated in Figures 5.10 and 5.11

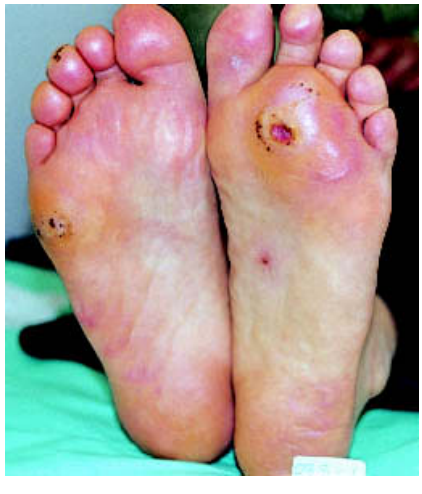

Figure 5.13 Healing neuropathic ulcers in the patient whose feet are shown in Figures 5.10–5.11. Note bunionette deformity at the right foot

right third metatarsal head a neuropathic ulcer was noted in an area of gross callus formation, in addition to claw toe deformity (Figure 5.11). A callus was present under her right fifth metatarsal head over a bunionette deformity. Mild callus formation was observed on the heels of both feet. Onychomycosis affecting all toes was also present (discussed in Chapter 8, see Figure 8.7).

A plain radiograph did not reveal osteomyelitis. Sharp debridement was performed and therapeutic half shoes were prescribed. In-shoe peak pressure measurement showed high pressures under both heels, metatarsal heads, and halluxes when the patient wore

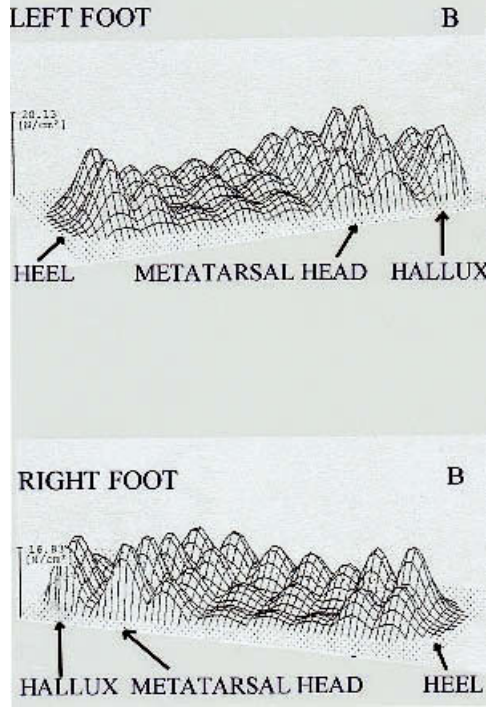

Figure 5.14 Effect of orthotic insoles and correct footwear on in-shoe peak plantar pressures on the left (upper panel) and right foot (lower panel) in the patient whose feet are illustrated in Figures 5.10–5.11

her own shoes (Figure 5.12). She had standard treatment on a weekly basis and the ulcers began to heal slowly. Six months after her first visit, an ulcer developed under her left third metatarsal head and a callus under her right fifth metatarsal head (Figure 5.13). New shoes were prescribed with orthotic insoles: the in-shoe peak pressures were reduced from 33.3 to 16.83 N/cm^2 under her right, and from 37.42 to 20.13 N/cm^2 under her left foot (Figure 5.14).

The patient continued visiting the outpatient foot clinic almost every week, and 6 months after her first visit her ulcers had healed.

Keywords: Neuropathic ulcer; peak plantar pressures

ULCERS OVER A CHARCOT FOOT

The following two figures (before and after debridement) show the left foot of a male patient of 62 years of age with type 2 diabetes diagnosed at the age of 48 years and treated with insulin. A smoker since the age of 18 years, the patient had had an ulcer on the plantar aspect of his left hallux which was complicated by osteomyelitis and led to amputation 3 years previously. One year before his first visit to the foot clinic the patient developed an ulcer on the lateral aspect of his left foot which resulted in osteomyelitis and surgical debridement of the metatarsal bone. After a femoral-popliteal bypass graft in his left foot, the patient developed neuro-osteoarthropathy. He presented to the outpatient clinic with two painless ulcers under his first and third metatarsal heads surrounded by hemorrhagic calluses. Hyperkeratosis under his fifth metatarsal head and a scar at the site of the surgical debridement were noted (Figure 5.15).

The graft was functioning well and the patient had no claudication. Debridement of the ulcer under his fourth metatarsal exposed the bone (Figure 5.16). Cultures were obtained from the sloughy base of the ulcer — a positive sign of infection — and the patient was treated with an empirical combination of cotrimoxazole and clindamycin. The patient did not attend follow-up, therefore no X-ray or any further studies are available.

Charcot foot typically does not develop in patients with peripheral vascular disease since increased blood supply to the bone is needed for the osseous tissue to be overmetabolized. Autonomic sympathetic neuropathy leads to bone arteriovenous

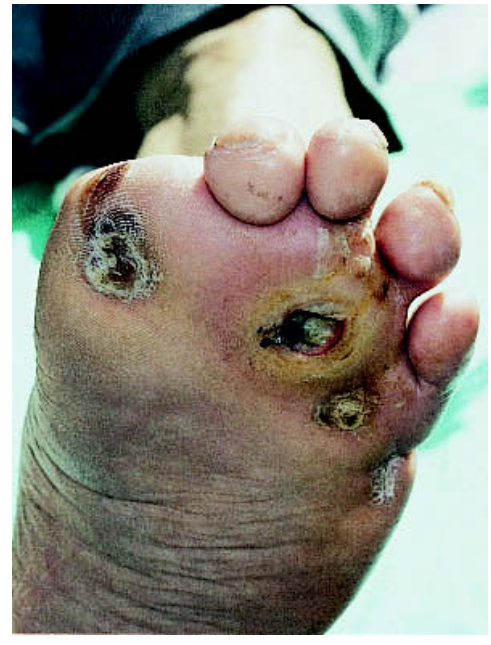

Figure 5.15 Hallux disarticulation at the metatarsophalangeal joint, callus under first and fifth metatarsal heads, and deep infected neuropathic ulcer under the third metatarsal head. Claw toes

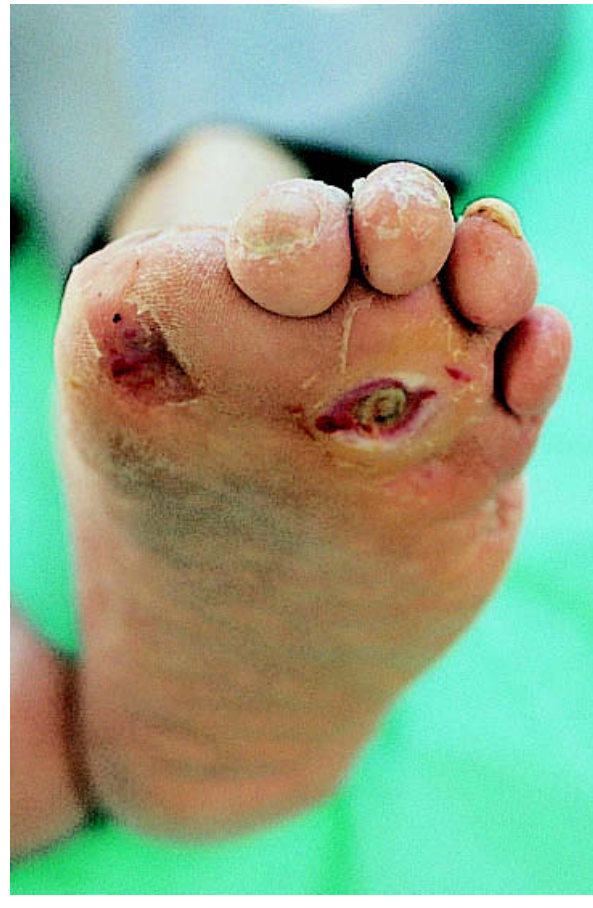

Figure 5.16 Foot shown in Figure 5.15 after sharp debridement. Note bone exposure at the base of the ulcer under the third metatarsal head

shunting, hypervascularity and demineralization. Some cases are reported to occur after bypass surgery of the arteries.

Exposure of the bone denotes osteomyelitis and it should be treated accordingly.

Keywords: Neuropathic ulcers; Charcot foot; osteomyelitis; amputation

A NEUROPATHIC ULCER UNDER THE HEEL

A 51-year-old female patient with type 2 diabetes since the age of 38 years and treated with insulin, was referred to the outpatient diabetic foot clinic because of a chronic non-healing ulcer under her right heel. She had good diabetes control (HBA_{1c}: 7.2%). Four months before her first visit she noticed a painless blister on the right heel caused by a small stone in her shoe; the blister ruptured and since the patient did not feel any pain she did not give her foot any attention. Some discharge was present on her socks, but it was the patient's daughter who saw a superficial ulcer on the right heel. The patient visited a primary care clinic and was advised to clean the ulcer with povidone iodide and apply clean dressings every day. A 2-week course of

antibiotics was prescribed. She continued her daily activities and after 4 months the ulcer was still active.

On examination the patient was found to have severe diabetic neuropathy with loss of sensation of pain, temperature, light touch and vibration. The vibration perception threshold was 36 V on both feet. Peripheral pulses were normal and the ankle brachial index was 1.2 and 1.1 in the right and left foot respectively. A full thickness ulcer with a sloughy base was noted on the right heel (Figure 5.17). No other signs of infection were present. An X-ray did not show involvement of the calcaneus. Cultures from the base of the ulcer revealed *Staphylococcus aureus*. She was treated with amoxicillin–clavulanic acid for 2 weeks and the ulcer was debrided on a weekly basis; dressings were changed daily. Meanwhile she was advised to rest and heel-free shoes to offload pressure from the ulcerated area were prescribed (Figure 5.18). After 6 months the ulcer had healed completely (Figure 5.19).

Bedridden patients develop heel ulcers or gangrene quite frequently (20–30%),

Figure 5.18 Commercially available heel-free shoes for the treatment of hindfoot ulcers

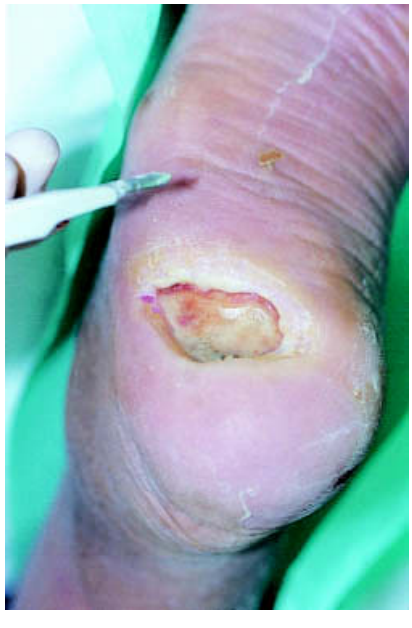

Figure 5.17 Deep heel neuropathic ulcer with infected sloughy bed caused by trauma

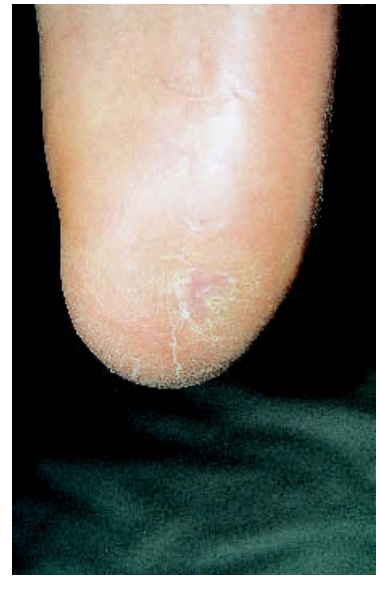

Figure 5.19 Hindfoot shown in Figure 5.17 after the ulcer has completely healed

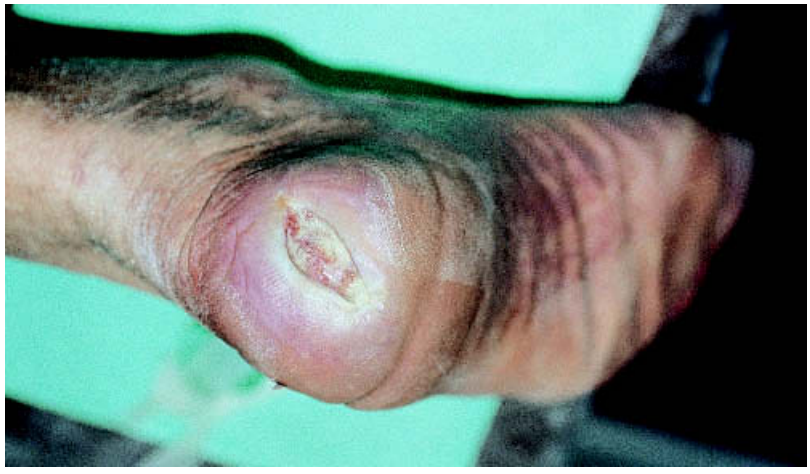

Figure 5.20 Neuropathic heel ulcer caused by shoe seam

usually on the posterolateral aspect. Excessive walking in new shoes can cause blister formation on the posterior aspect of the heel in patients with neuropathy. Shoe seams may also cause ulcers on the heel (Figure 5.20). Therefore shoes and socks without seams are prescribed to patients with loss of protective sensation. Heel ulceration is difficult in management since debridement in this area precludes functional weight bearing. Major amputations are often necessary when heel ulcers are infected.

Keywords: Neuropathic ulcer; heel

BURNS ON TOES AND FOREFOOT

A 55-year-old male patient with type 2 diabetes since the age of 43 years attended the outpatient diabetic foot clinic due to ulcers on his feet. His diabetes was poorly controlled with sulfonylureas and he had a history of a disarticulated left great toe at the metatarsophalangeal joint due to osteomyelitis.

On examination the patient was febrile; peripheral pulses were palpable, the ankle brachial index was 1.2; the vibration perception threshold was over 50 V in both feet; temperature, light touch and pinprick sensation were absent as were the Achilles tendon reflexes. Blood pressure was normal; no other diabetic complications were found. HbA_{1c} was 11.0%. There was a perforating dirty ulcer on the outer aspect of his right foot. A large amount of callus had built up around the plantar orifice (Figures 5.21 and 5.22). The patient reported edema of the forefoot which had recently subsided as was evident from the scaling of the skin. Callus formation was also observed over the second, third and fifth metatarsal heads of the left foot. The patient was empirically treated with ciprofloxacin.

Debridement of the callus was carried out. Cultures revealed *Staphylococcus aureus and Escherichia coli.* Osteomyelitis of the fifth metatarsal head was evident on a plain radiograph (Figure 5.23). The patient

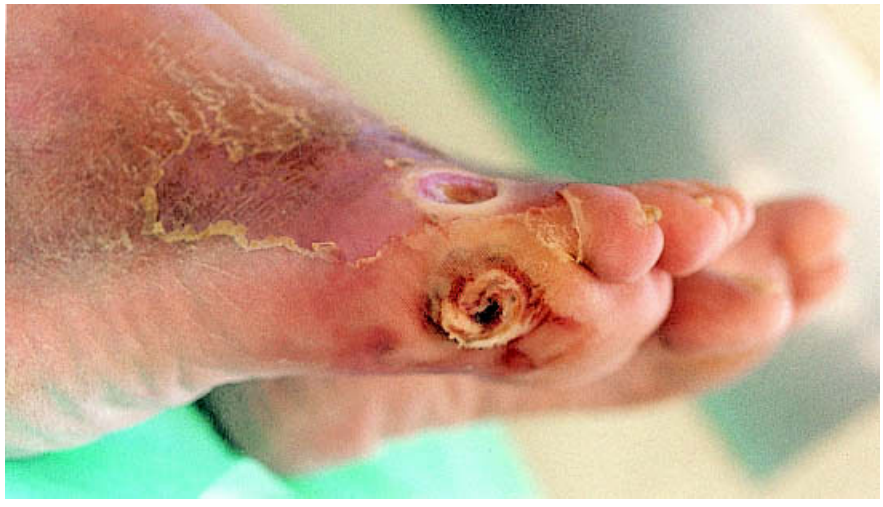

Figure 5.21 Perforating, infected neuropathic ulcer under the fifth metatarsal head. Scaling is due to edema that has subsided

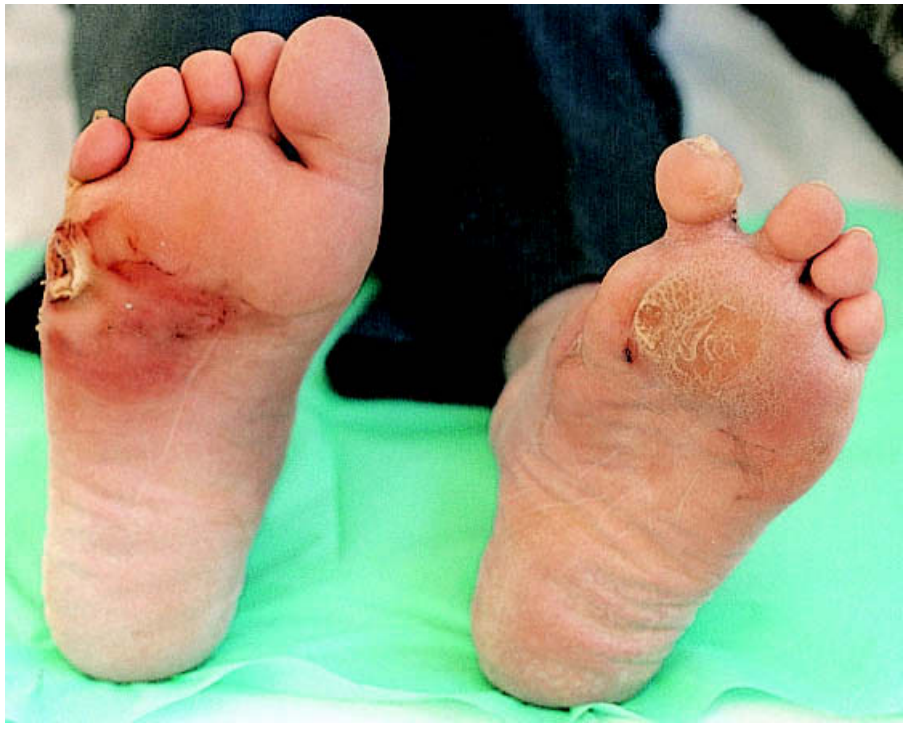

Figure 5.22 Right foot: neuropathic ulcer shown in Figure 5.21. Left foot: hallux disarticulation, medial displacement of second toe with claw deformity; callus formation under second, third and fifth metatarsal heads

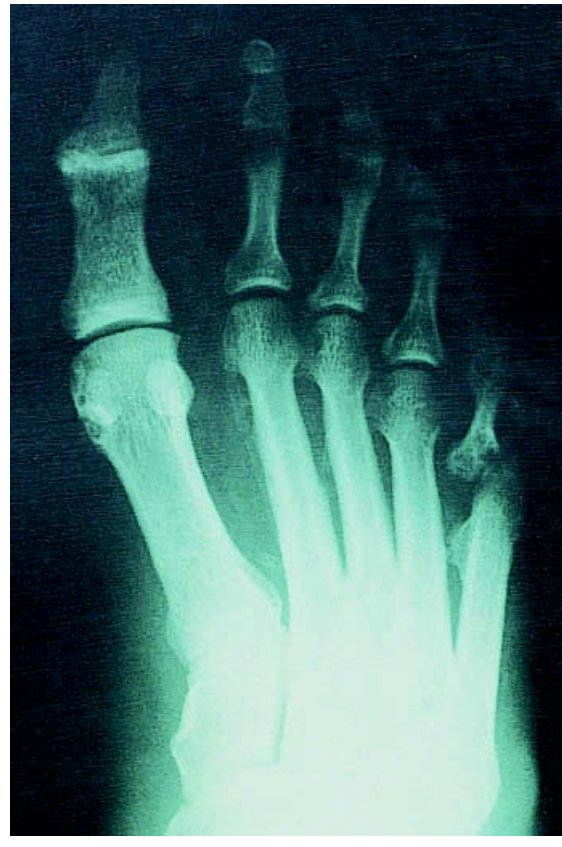

Figure 5.23 Plain radiograph of the right foot of the patient whose foot is shown in Figure 5.21. Osteomyelitis of the fifth metatarsal head and the proximal phalanx of the fifth toe, subluxation of the metatarsophalangeal joint, calcification of the digital artery between the first two metatarsals and osteoarthritis of the first distal phalangophalangeal joint of the hallux are all apparent

continued ciprofloxacin treatment; cotrimoxazole was added for almost 6 months and the ulcer gradually healed (Figure 5.24) with the help of therapeutic shoes.

Instruction in appropriate foot care was provided. The patient visited the outpatient clinic erratically; callus formation on the site of the healed ulcer was removed every 3 months; he refused strict glycemic control as he was afraid that episodes of hypoglycemia would jeopardize his position at work. He used intermediate-acting insulin at bedtime and sulfonylureas during the day. His HbA_{1c} remained at 9.0% during the following year. Preventive footwear was not accepted.

The patient attended the clinic 2 years later because of multiple burns over the tips of his toes and superficial ulcers over the fifth metatarsal heads of both feet (Figure 5.25). He had put his feet in front of the fire in order to dry out his wet socks. No pain was felt. Although the patient was aware of the burns he continued his activities for a week before this visit.

Full thickness burns were present over the tips of all toes. Blisters over the right fifth metatarsal head and the left fourth and fifth toes were removed and ulcers had developed since the patient was still working regularly, despite medical advice to the contrary (Figure 5.26). Calluses formed around the new plantar ulcers. Amoxicillin–clavulanic acid treatment was initiated and the patient attended the diabetic foot clinic on a weekly basis.

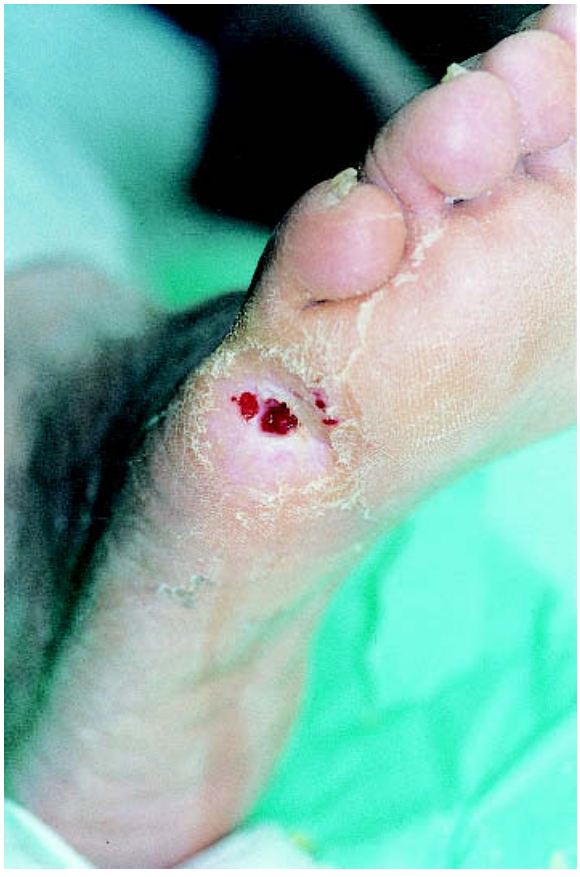

Figure 5.24 The ulcer shown in Figure 5.21 after it has almost completely healed

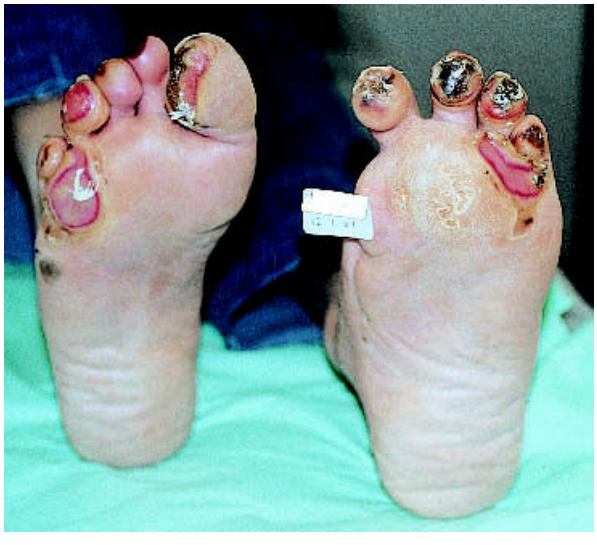

Figure 5.25 Thermal injury sustained by the patient whose feet are illustrated in Figure 5.22

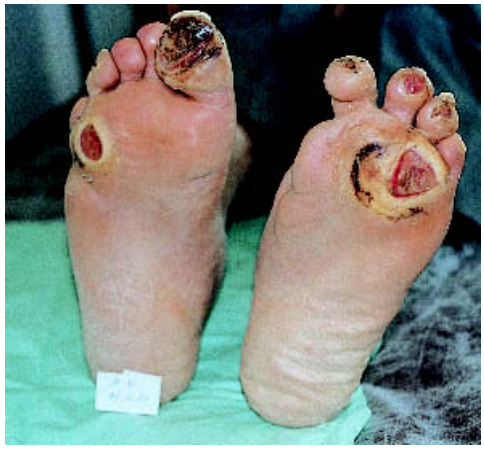

Figure 5.26 Neuropathic ulcers under the fifth metatarsal heads and progression of thermal injury in the patient whose feet are shown in Figures 5.21–5.25. The patient did not comply with doctors' instructions

All ulcers healed within 2 months except the one on the right great toe, which was complicated by osteomyelitis and acute soft tissue infection. Five months after the burn his right hallux had to be disarticulated.

The patient still refused preventive shoes and 4 months after this second amputation new ulcers developed under the fifth metatarsal heads bilaterally (Figure 5.27).

Keywords: Thermal injury; osteomyelitis

CHRONIC NEUROPATHIC ULCER COMPLICATED BY OSTEOMYELITIS

A 55-year-old male patient with type 2 diabetes diagnosed at the age of 50 years was referred to the outpatient diabetic foot clinic because of a chronic neuropathic ulcer on his right foot. He had a history of hypertension, background retinopathy in both eyes and diabetic nephropathy (urine protein 1.5 g/24 h). He had been treated with sulfonylurea but had discontinued

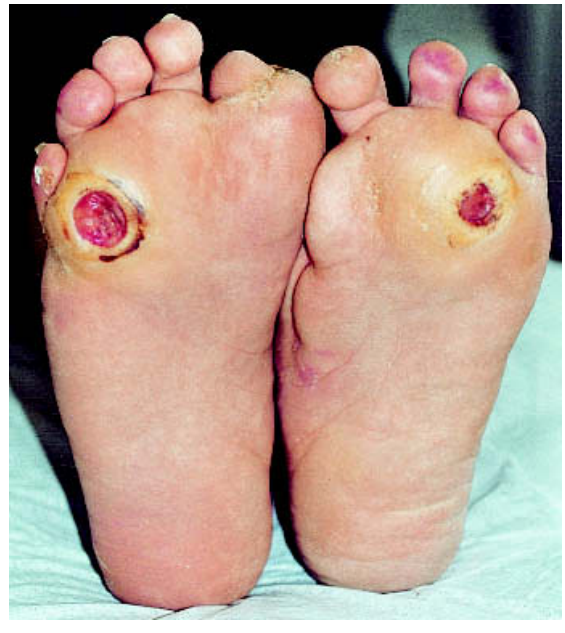

Figure 5.27 Right hallux disarticulation at the metatarsophalangeal joint and recurrence of ulcers under the fifth metatarsal heads (patient whose feet are shown in Figures 5.21–5.26)

the treatment 1 year before his first visit, when overt nephropathy developed. He had excellent diabetes control (HBA_{1c}: 6.4%).

On examination his feet pulses were bounding (ankle pressure index 1.2 bilaterally); he had severe peripheral neuropathy: no sensation of pain, light touch, vibration or temperature; the vibration perception threshold was 48 V on the left and above 50 V on the right foot. A full thickness clear neuropathic ulcer surrounded by callus was observed under the right first metatarsal head, with dimensions of 3 × 3 × 0.5 cm (Figure 5.28). Mild claw deformities of the toes and displacement of the metatarsal fat pads to the base of the proximal phalanges due to muscle atrophy were also noted.

Sharp debridement was carried out and special extra depth shoes with an orthotic insole were prescribed. Care was taken to offload pressure from the ulcerated area. The patient was advised to limit his daily activities and he attended the diabetes foot clinic on a weekly basis. The size of the ulcer was reduced by half within 4 weeks. Two weeks later, after a professional trip, the patient visited the clinic again. His ulcer was infected and a large amount of callus had formed around it. His right hallux had a 'sausage-like' appearance and signs of infection were observed (redness and edema). A culture from the base of the ulcer revealed the presence of *Staphylococcus aureus* and *Enterobacter cloacae* post-debridement. A radiograph at that time showed mild erosion of the first metatarsal head.

The patient was given treatment with cotrimoxazole and clindamycin. The radiograph was repeated 2 weeks later and extensive erosion of the first metatarsal head was revealed (Figure 5.29). Acute osteomyelitis was diagnosed. The patient continued with the antibiotics for 12 weeks and had regular chiropody treatment on a weekly basis. The ulcer healed completely in 20 weeks (Figure 5.30).

Treatment of acute osteomyelitis should be based on bone cultures when possible, and should be continued for 6–12 weeks. The commonest pathogen of

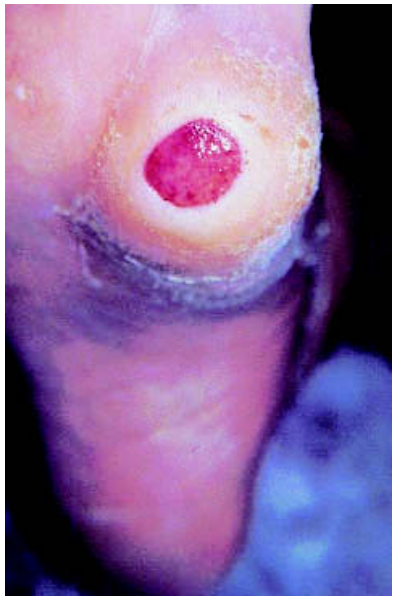

Figure 5.28 Neuropathic ulcer under prominent first metatarsal head. Healthy granulating tissue can be seen at the base of the ulcer

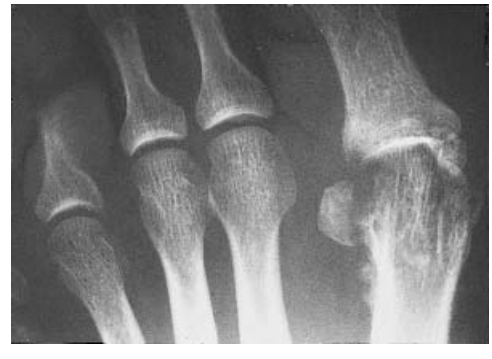

Figure 5.29 Erosion of first metatarsal head with periosteal reaction due to osteomyelitis (patient whose ulcer is shown in Figure 5.28)

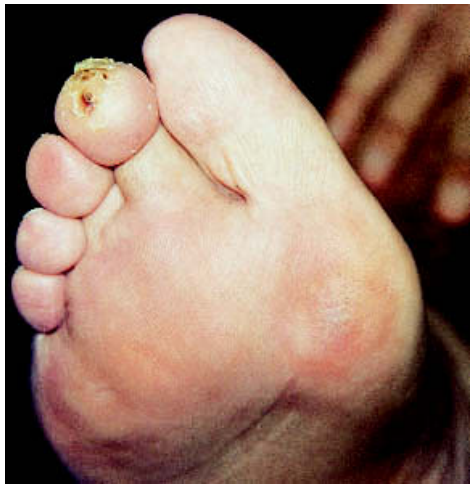

Figure 5.30 Healed ulcer of the patient whose feet are shown in Figures 5.28–5.29. Note the scar over the ulcerated area and callus formation at the tip of the second toe due to claw deformity

acute osteomyelitis in patients with foot ulcers is *Staphylococcus aureus* (60–90%). Other pathogens include *Staphylococcus epidermidis*, *Escherichia coli*, *Pseudomonas aeruginosa*, and other *Enterobacter* spp. More than one pathogen is often isolated. In order to achieve therapeutic levels of antibiotics in the bone it is preferable to administer antibiotics intravenously for the first 2 weeks. However, oral antibiotics with good bioavailability (fluoroquinolones, clindamycin) may be adequate for therapy. Treatment regimens for staphylococcal osteomyelitis are as follows:

- Clindamycin 600 mg × 3 orally or 600 mg × 3 i.v.
- Fucidic acid 500 mg × 3 orally or 500 mg × 3 in a 500-ml solution delivered slowly i.v. (over 4–6 h)
- Cotrimoxazole 960 mg × 2 orally or i.v.
- Ciprofloxacin 750 mg × 2 orally, or 400 mg × 3 i.v.
- Rifampicin 900 mg × 1 orally or i.v.
- Teicoplanin 600 mg × 1 orally or i.m. or i.v.
- Vancomycin 500 mg × 4 i.v. or 1 g × 2 i.v.

Fluoroquinolones, teicoplanin and vancomycin should be prescribed for methicillin-resistant staphylococcus only. Fluoroquinolones in particular, should always be combined with another anti-staphylococcal drug in the first month of treatment, since it is likely that a resistant strain will prevail in the infection.

Keywords: Acute osteomyelitis; treatment